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L1: Entry 1 of 7

File: PGPB

Aug 14, 2003

PGPUB-DOCUMENT-NUMBER: 20030154032

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030154032 A1

TITLE: Methods and compositions for diagnosing and treating rheumatoid arthritis

PUBLICATION-DATE: August 14, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Pittman, Debra D.	Windham	NH	US	
Feldman, Jeffrey L.	Arlington	MA	US	
Shields, Kathleen M.	Harvard	MA	US	
Trepicchio, William L.	Andover	MA	US	

US-CL-CURRENT: 702/20

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KWIC
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☐ 2. Document ID: US 20030143552 A1

L1: Entry 2 of 7

File: PGPB

Jul 31, 2003

PGPUB-DOCUMENT-NUMBER: 20030143552

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030143552 A1

TITLE: Novel compositions and methods for the identification, assessment, prevention and therapy of human cancers

PUBLICATION-DATE: July 31, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Clark, Edwin	Ashland	MA	US	
Grenfell-Lee, Tallessyn	Cambridge	MA	US	
Lu, Karen	Houston	TX	US	
Hartmann, Lynn	Rochester	MN	US	
Brown, Jeffrey L.	Arlington	MA	US	

US-CL-CURRENT: [435/6](#); [435/7.23](#)

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KWIC
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☐ 3. Document ID: US 20030105594 A1

L1: Entry 3 of 7

File: PGPB

Jun 5, 2003

PGPUB-DOCUMENT-NUMBER: 20030105594

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030105594 A1

TITLE: cDNA databases for analysis of hematopoietic tissue

PUBLICATION-DATE: June 5, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Westbrook, Carol A.	Chicago	IL	US	
Hoffman, Ronald	Chicago	IL	US	

US-CL-CURRENT: [702/19](#); [702/20](#)

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KWIC
Draw Desc	Image										

☐ 4. Document ID: US 20030040089 A1

L1: Entry 4 of 7

File: PGPB

Feb 27, 2003

PGPUB-DOCUMENT-NUMBER: 20030040089

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030040089 A1

TITLE: Protein-protein interactions in adipocyte cells

PUBLICATION-DATE: February 27, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Legrain, Pierre	Paris		FR	
Marullo, Stefano	Paris		FR	
Ralf, Jockers	Bures Sur Yvette		FR	

US-CL-CURRENT: [435/183](#); [435/320.1](#), [435/325](#), [435/69.1](#), [435/7.1](#), [536/23.2](#), [702/19](#)

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KWIC
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☐ 5. Document ID: US 20030036070 A1

L1: Entry 5 of 7

File: PGPB

Feb 20, 2003

PGPUB-DOCUMENT-NUMBER: 20030036070

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030036070 A1

TITLE: Gene expression profiling of inflammatory bowel disease

PUBLICATION-DATE: February 20, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Chakravarti, Shukti	Lutherville	MD	US	

US-CL-CURRENT: [435/6](#); [435/91.2](#)

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments		KWIC
Draw Desc	Image										

☐ 6. Document ID: US 20020142327 A1

L1: Entry 6 of 7

File: PGPB

Oct 3, 2002

PGPUB-DOCUMENT-NUMBER: 20020142327

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020142327 A1

TITLE: Expression analysis of smarc nucleic acids and polypeptides useful in the diagnosis and treatment of prostate cancer

PUBLICATION-DATE: October 3, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Gillis, Kimberly A.	Swampscott	MA	US	
Zhang, Yixian	Pearl River	NY	US	

US-CL-CURRENT: 435/6; 435/7.23, 514/1

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	KWIC
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☐ 7. Document ID: NO 200302394 A WO 200244420 A2 AU 200227004 A US 20020142327 A1

L1: Entry 7 of 7

File: DWPI

Jul 2, 2003

DERWENT-ACC-NO: 2002-537461

DERWENT-WEEK: 200356

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TITLE: Assessing if a subject is afflicted with prostate cancer by comparing the expression level of SWI/SNF-related matrix-associated actin-dependent regulator of chromatin marker in sample from a subject and control sample

INVENTOR: GILLIS, K A; ZHANG, Y

PRIORITY-DATA: 2000US-253487P (November 28, 2000), 2001US-0997424 (November 28, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
NO 200302394 A	July 2, 2003		000	C12Q001/68
WO 200244420 A2	June 6, 2002	E	095	C12Q001/68
AU 200227004 A	June 11, 2002		000	C12Q001/68
US 20020142327 A1	October 3, 2002		000	C12Q001/68

INT-CL (IPC): A61 K 31/00; C12 Q 1/68; G01 N 33/574

ABSTRACTED-PUB-NO: WO 200244420A

BASIC-ABSTRACT:

NOVELTY - Assessing (M1) if subject is afflicted with prostate cancer, involves comparing expression level of SWI/SNF-related matrix-associated actin-dependent regulator of chromatin (SMARC) marker (I) in sample (S) from subject, and normal expression level of (I) in control sample (CS), where significant difference between level of expression of (I) in (S) and CS indicates that the subject is afflicted with cancer.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) monitoring (M2) the progression of prostate cancer in a subject, by detecting in a subject sample at a first point in time, the expression of a marker selected from one or more SMARCD3 markers or their combination, repeating the above mentioned step at a subsequent point in time and comparing the level of expression detected in the above mentioned steps, and monitoring the progression of prostate cancer in the subject;

(2) assessing (M3) the efficacy of a therapy for inhibiting prostate cancer in a subject, by comparing expression of SMARCD3 marker in a first sample obtained from the subject prior to providing at least a portion of the therapy to the subject, and the expression of the SMARCD3 marker in a second sample obtained from the subject following provision of the portion of the therapy, where a significantly enhanced level of expression of the marker in the second sample, relative to the first sample is an indication that the therapy is efficacious for inhibiting prostate cancer in the subject;

(3) assessing (M4) the potential of a test compound to trigger prostate cancer in a cell, by maintaining separate aliquots of cells in the presence and absence of the test compound, and comparing expression of a SMARCD3 marker in each of the aliquots, where a significantly reduced level of expression of the marker in the aliquot maintained in the presence of the test compound, relative to the aliquot maintained in the absence of the test compound, is an indication that the test compound possesses the potential for triggering prostate cancer in a cell;

(4) treating (M1) a subject afflicted with prostate cancer, by administering a compound which increases the expression of a SMARCD3 marker;

(5) identifying (M1) a compound useful for treating prostate cancer by:

(a) measuring the expression level of a SMARCD3 marker, comparing the expression measured to the expression of the marker in a cell in the absence of the test compound, where the compound is useful for treating prostate cancer when the expression level of SMARCD3 in the presence of the test compound is higher than its expression level in the absence of the test compound; or

(b) measuring an activity of a SMARCD3 marker, comparing the activity measured to the level of activity of the marker in the absence of the test compound, where the compound is useful for treating prostate cancer when the activity of SMARCD3 marker in the presence of the test compound is higher than its activity in the absence of the test compound;

(6) determining (M1) the efficacy of androgen withdrawal treatment in a subject afflicted with prostate cancer, by detecting in a subject sample at a first point in time, the expression level of a SMARCD3 marker, repeating the above mentioned step at a subsequent point in time occurring after the subject begins androgen withdrawal treatment, and comparing the level of expression of markers detected in the above mentioned steps, where an increase in the level of expression indicates that the androgen withdrawal treatment has reduced efficacy; and

(7) inhibiting (M1) prostate cancer in a subject by inhibiting expression of SMARCD3 marker in the cells of a subject.

ACTIVITY - Cytostatic.

No suitable data given.

MECHANISM OF ACTION - None given.

USE - (M1) is useful for assessing if a subject is afflicted with prostate cancer. (M1) is useful for treating a subject afflicted with prostate cancer (claimed).

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	KWIC
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L1: Entry 2 of 7

File: PGPB

Jul 31, 2003

PGPUB-DOCUMENT-NUMBER: 20030143552

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030143552 A1

TITLE: Novel compositions and methods for the identification, assessment, prevention and therapy of human cancers

PUBLICATION-DATE: July 31, 2003

US-CL-CURRENT: 435/6; 435/7.23APPL-NO: 10/ 071510 [PALM]

DATE FILED: February 8, 2002

RELATED-US-APPL-DATA:

Application is a non-provisional-of-provisional application 60/267276, filed February 8, 2001,

RELATED APPLICATIONS

[0001] This application claims priority to U.S. Provisional Application No. 60/267,276 filed on Feb. 8, 2001, the contents of which are incorporated herein by reference.

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L1: Entry 6 of 7

File: PGPB

Oct 3, 2002

PGPUB-DOCUMENT-NUMBER: 20020142327

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020142327 A1

TITLE: Expression analysis of smarc nucleic acids and polypeptides useful in the diagnosis and treatment of prostate cancer

PUBLICATION-DATE: October 3, 2002

US-CL-CURRENT: 435/6; 435/7.23, 514/1

APPL-NO: 09/ 997424 [PALM]

DATE FILED: November 28, 2001

RELATED-US-APPL-DATA:

Application is a non-provisional-of-provisional application 60/253487, filed November 28, 2000,

RELATED APPLICATION

[0001] This application claims the benefit of U.S. Provisional Patent Application Serial No. 60/253,487, filed Nov. 28, 2000, entitled "Expression Analysis of SMARC Nucleic Acids and Polypeptides Useful in the Diagnosis and Treatment of Prostate Cancer". The teachings of the foregoing application is incorporated herein by reference.

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L1: Entry 6 of 7

File: PGPB

Oct 3, 2002

PGPUB-DOCUMENT-NUMBER: 20020142327

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020142327 A1

TITLE: Expression analysis of smarc nucleic acids and polypeptides useful in the diagnosis and treatment of prostate cancer

PUBLICATION-DATE: October 3, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Gillis, Kimberly A.	Swampscott	MA	US	
Zhang, Yixian	Pearl River	NY	US	

US-CL-CURRENT: 435/6; 435/7.23, 514/1

CLAIMS:

What is claimed:

1. A method of assessing whether a subject is afflicted with prostate cancer, comprising: a) obtaining a level of expression of a marker in a sample from said subject, wherein said marker is selected from the group consisting of one or more SMARC markers; b) obtaining a normal level of expression of said marker in a control sample; and c) comparing (a) with (b), wherein a significant difference between said level of expression of said marker in said sample from said subject, and said normal level is an indication that said subject is afflicted with prostate cancer.
2. The method of claim 1, wherein said marker corresponds to a transcribed polynucleotide or portion thereof.
3. The method of claim 1, wherein said sample comprises cells obtained from said subject.
4. The method of claim 3, wherein said cells are collected from a prostate gland.
5. The method of claim 3, wherein said cells are collected from blood.
6. The method of claim 1, wherein said level of expression of said marker in said sample differs from said normal level of expression of said marker in a subject not afflicted with prostate cancer by a factor of about at least 2.
7. The method of claim 1, wherein said level of expression of said marker in said sample differs from said

normal level of expression of said marker in a subject not afflicted with prostate cancer by a factor of above at least 3.

8. The method of claim 1, wherein said level of expression of said marker in said sample is assessed by detecting the presence in said sample of a protein corresponding to said marker.

9. The method of claim 8, wherein said presence of said protein is detected using a reagent which specifically binds with said protein.

10. The method of claim 9, wherein said reagent is selected from the group consisting of an antibody, an antibody derivative, and an antibody fragment.

11. The method of claim 1, wherein said level of expression of said marker in said sample is assessed by detecting the presence in said sample of a transcribed polynucleotide or portion thereof.

12. The method of claim 11, wherein said transcribed polynucleotide is a mRNA.

13. The method of claim 11, wherein said transcribed polynucleotide is a cDNA.

14. The method of claim 11, wherein said detecting the presence in said sample of a transcribed polynucleotide or portion thereof further comprises amplifying said transcribed polynucleotide or portion thereof.

15. The method of claim 1, wherein said level of expression of said marker in said sample is assessed by detecting the presence in said sample of a transcribed polynucleotide, or portion thereof, which anneals with said marker, or portion thereof, under stringent hybridization conditions.

16. The method of claim 1, further comprising comparing: a) the level of expression in said sample of at least two SMARC markers independently, and b) the normal level of expression of each of at least two SMARC markers in samples of the same type obtained from control subjects not afflicted prostate cancer, c) comparing (a) with (b), wherein said level of expression of more than one of the markers is significantly altered, relative to the corresponding normal levels of expression of the markers, is an indication that the subject is afflicted prostate cancer.

17. A method for monitoring the progression of prostate cancer in a subject, the method comprising: a) detecting in a subject sample at a first point in time, the expression of a marker, wherein the marker is selected from the group consisting of one or more SMARC markers or a combination thereof; b) repeating step (a) at a subsequent point in time; and c) comparing said level of expression detected in steps (a) and (b), and therefrom monitoring the progression of prostate cancer in said subject.

18. The method of claim 17, wherein said marker corresponds to a transcribed polynucleotide or portion thereof.

19. The method of claim 17, wherein the sample comprises cells obtained from said subject.

20. The method of claim 19, wherein said cells are collected from a prostate gland.

21. The method of claim 19, wherein said cells are collected from blood.

22. A method of assessing the efficacy of a therapy for inhibiting prostate cancer in a subject, comprising:

a) expression of a SMARCD3 marker in a first sample obtained from said subject prior to providing at least a portion of said therapy to said subject; b) expression of the SMARCD3 marker in a second sample obtained from said subject following therapeutic treatment; and c) comparing (a) with (b), wherein a significantly enhanced level of expression of said marker in said second sample, relative to said first sample, is an indication that said therapy is efficacious for inhibiting prostate cancer in said subject.

23. A method of assessing the potential of a test compound to trigger prostate cancer in a cell, comprising: a) maintaining separate aliquots of cells in the presence and absence of said test compound; and b) comparing expression of a RCD3 marker in each of said aliquots, wherein a significantly reduced level of expression of the marker in the aliquot maintained in the presence of the test compound, relative to said aliquot maintained in the absence of said test compound, is an indication that said test compound possesses the potential for triggering prostate cancer in a cell.

24. A method of inhibiting prostate cancer in a subject at risk for developing prostate cancer, the method comprising inhibiting expression of SMARCD3 marker in the cells of a subject.

25. A method for identifying a compound useful for treating prostate cancer, comprising: a) measuring the expression level of a SMARCD3 marker in a cell in the presence of a test compound; and b) comparing the expression measured in step (a) to the expression of the SMARCD3 marker in a cell in the absence of said test compound, wherein said compound is useful for treating prostate cancer when the expression level of said marker in the presence of said test compound is higher than its expression level in the absence of said test compound.

26. The method of claim 25, wherein said expression level is determined by measuring the levels of mRNA of said marker.

27. The method of claim 25 wherein said expression level is determined by measuring the levels of protein of said marker.

28. A method for identifying a compound useful for treating prostate cancer, comprising a) measuring an activity of a SMARDCD3 marker; and b) comparing the activity measured in step (a) to the level of activity of said marker in the absence of the test compound, wherein said compound is useful for treating prostate cancer when the activity of the SMARCD3 marker in the presence of said test compound is higher than its activity in the absence of said test compound.

29. The method of claim 28, wherein said cell is a prostate cancer cell.

30. A method of treating prostate cancer in a patient, comprising administering to the patient in need thereof a compound which increases the expression of a SMARCD3 marker.

31. The method of claim 30, wherein said compound increases the expression of a SMARCD3 mRNA.

32. The method of claim 31, wherein said compound decreases expression of a SMARCD3 marker protein.

33. A method for determining the efficacy of androgen withdrawal treatment in a subject afflicted with prostate cancer, comprising: a) detecting in said subject sample at a first point in time, the expression level of a SMARCD3 marker; b) repeating step (a) at a subsequent point in time occurring after said subject begins androgen withdrawal treatment; and c) comparing the level of expression of said marker detected in steps (a) and (b), wherein a increase in said level of expression indicates that the androgen withdrawal treatment has reduced efficacy.

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File 155:MEDLINE(R) 1966-2003/Nov W3
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*File 155: Medline has temporarily stopped updating with
Completed records (Nov 2003). Please see HELP NEWS 154 for details.
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File 34:SciSearch(R) Cited Ref Sci 1990-2003/Nov W3
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File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
      (c) 1998 Inst for Sci Info
File 340:CLAIMS(R)/US Patent 1950-03/Nov 18
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*File 340: Enter HELP NEWS340 & HELP ALERTS340 for search,
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11476260 98360103 PMID: 9693044
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Five SWI/SNF-related, matrix-associated, actin-dependent regulator of chromatin (SMAR) genes are dispersed in the human genome.

Ring H Z; Vameghi-Meyers V; Wang W; Crabtree G R; Francke U

Department of Genetics, Stanford University School of Medicine, Stanford, California, 94305, USA.

Genomics (UNITED STATES) Jul 1 1998, 51 (1) p140-3, ISSN 0888-7543
Journal Code: 8800135

Contract/Grant No.: GM08408; GM; NIGMS; HG00298; HG; NHGRI; NS10447; NS;
NINDS

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

The SWI/SNF-related, matrix-associated, actin-dependent regulators of chromatin (SMAR), also called BRG1-associated factors, are components of human SWI/SNF-like chromatin-remodeling protein complexes. We mapped five human SMAR genes to regions on four different human chromosomes, SMARCC1 to 3p23-p21, SMARCC2 to 12q13-q14, **SMARCD1** to 12q13-q14, **SMARCD2** to 17q23-q24, and **SMARCD3** to 7q35-q36. SMARCC1, SMARCC2, and **SMARCD1** are assigned to chromosomal regions that are frequently involved in somatic rearrangements in human cancers. **SMARCD1** was mapped to the critical region of Allgrove syndrome; however, no mutation was identified in one Allgrove syndrome family studied. Copyright 1998 Academic Press.

... genes to regions on four different human chromosomes, SMARCC1 to 3p23-p21, SMARCC2 to 12q13-q14, **SMARCD1** to 12q13-q14, **SMARCD2** to 17q23-q24, and **SMARCD3** to 7q35-q36. SMARCC1, SMARCC2, and **SMARCD1** are assigned to chromosomal regions that are frequently involved in somatic rearrangements in human cancers. **SMARCD1** was mapped to the critical region of Allgrove syndrome; however, no mutation was identified in...

1/3,K,AB/2 (Item 1 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)
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0014356691 BIOSIS NO.: 200300315410
MICROARRAY ANALYSIS OF SEX DIFFERENCES IN THE MOUSE CNS TRANSCRIPTOME.
AUTHOR: Chesler E J (Reprint); Shou S (Reprint); Qu Y (Reprint); Yang X (Reprint); Lu L (Reprint); Williams R W (Reprint)
AUTHOR ADDRESS: Department of Anatomy and Neurobiology, Center of Genomics and Bioinformatics, University of Tennessee Health Science Center, Memphis, TN, USA**USA
JOURNAL: Society for Neuroscience Abstract Viewer and Itinerary Planner
2002 pAbstract No. 623.6 2002 2002
MEDIUM: cd-rom
CONFERENCE/MEETING: 32nd Annual Meeting of the Society for Neuroscience
Orlando, Florida, USA November 02-07, 2002; 20021102
SPONSOR: Society for Neuroscience
DOCUMENT TYPE: Meeting; Meeting Poster; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Microarray transcript profiling provides a rapid and effective way to evaluate sex differences in the expression of many mRNAs in brain. We screened samples of forebrain, cerebellum, hippocampus, brainstem, olfactory bulb, eye, and liver from sets of sexually mature male and female mice using Affy U74Av2 arrays. Relative differences in expression level (i.e. fold changes) are often used to contrast expression between groups. The choice of thresholds is arbitrary and subject to high error rates because information on sample variance is not exploited. Here we examine the relation between inferential statistics and fold change thresholds. Using sample sizes sufficient to provide reasonable power (n = 14 male arrays, 59 female arrays, 3 cases per array), even an apparently stringent 2X threshold gives high error rates. The power of parametric analysis with adequate sample size is demonstrated by the detection of an appreciable number of transcripts with small yet significant differences. For use by other investigators we provide a complete list of expression profiles for 12422 transcripts (www.nervenet.org/papers/brainsexdifferences.html). The following short list of transcripts has significantly higher expression in female forebrain: Xist, Atp5g2, Cldn5, Eif2s3x, Rps19, Ptpn16, Rpl13a, and Sgk; whereas the following have higher expression in males: Dby, Eif2s3y, Gpcr12, **Smarcd2**, Crybb2, Tsga2, Bzrp, Csnk2b, Olfr37b, Adam5, Ednrb, and Krtap9-1. Expression differences in Dby, Xist, and several other transcripts were confirmed by quantitative rtPCR.

...ABSTRACT: Ptpn16, Rpl13a, and Sgk; whereas the following have higher expression in males: Dby, Eif2s3y, Gpcr12, **Smarcd2**, Crybb2, Tsga2, Bzrp, Csnk2b, Olfr37b, Adam5, Ednrb, and Krtap9-1. Expression differences in Dby, Xist...

1/3,K,AB/3 (Item 2 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

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0011597614 BIOSIS NO.: 199800391861

Five SWI/SNF-related, matrix-associated, actin-dependent regulator of chromatin (SMARCC) genes are dispersed in the human genome

AUTHOR: Ring Huijun Z; Vameghi-Meyers Vida; Wang Weidong; Crabtree Gerald R ; Francke Uta (Reprint)

AUTHOR ADDRESS: Howard Hughes Med. Inst., Stanford Univ. Sch. Med., Stanford, CA 94305-5323, USA**USA

JOURNAL: Genomics 51 (1): p140-143 July 1, 1998 1998

MEDIUM: print

ISSN: 0888-7543

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: The SWI/SNF-related, matrix-associated, actin-dependent regulators of chromatin (SMARCC), also called BRG1-associated factors, are components of human SWI/SNF-like chromatin-remodeling protein complexes. We mapped five human SMARCC genes to regions on four different human chromosomes, SMARCC1 to 3p23-p21, SMARCC2 to 12q13-q14, **SMARCD1** to 12q13-q14, **SMARCD2** to 17q23-q24, and **SMARCD3** to 7q35-q36. SMARCC1, SMARCC2, and **SMARCD1** are assigned to chromosomal regions that are frequently involved in somatic rearrangements in human cancers. **SMARCD1** was mapped to the critical region of Allgrove syndrome; however, no mutation was identified in one Allgrove syndrome family studied.

...ABSTRACT: to regions on four different human chromosomes, SMARCC1 to 3p23-p21, SMARCC2 to 12q13-q14, **SMARCD1** to 12q13-q14, **SMARCD2** to 17q23-q24, and **SMARCD3** to 7q35-q36. SMARCC1, SMARCC2, and **SMARCD1** are assigned to chromosomal regions that are frequently involved in somatic rearrangements in human cancers. **SMARCD1** was mapped to the critical region of Allgrove syndrome; however, no mutation was identified in...

DESCRIPTORS:

CHEMICALS & BIOCHEMICALS: ...**SMARCD1** gene...

...**SMARCD2** gene...

...**SMARCD3** gene

1/3,K,AB/4 (Item 1 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

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06949242 Genuine Article#: 106ZU Number of References: 19

Title: Five SWI/SNF-related, matrix-associated, actin-dependent regulator of chromatin (SMARCC) genes are dispersed in the human genome (ABSTRACT AVAILABLE)

Author(s): Ring HZ; VameghiMeyers V; Wang WD; Crabtree GR; Francke U (REPRINT)

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Abstract: The SWI/SNF-related, matrix-associated, actin-dependent regulators of chromatin (SMARC), also called BRG1-associated factors, are components of human SWZ/SNF-like chromatin-remodeling protein complexes. We mapped five human SMARC genes to regions on four different human chromosomes, SMARCC1 to 3p23-p21, SMARCC2 to 12q13-q14, **SMARCD1** to 12q13-q14, **SMARCD2** to 17q23-q24, and **SMARCD3** to 7q35-q36. SMARCC1, SMARCC2, and **SMARCD1** are assigned to chromosomal regions that are frequently involved in somatic rearrangements in human cancers. **SMARCD1** was mapped to the critical region of Allgrove syndrome; however, no mutation was identified in one Allgrove syndrome family studied. (C) 1998 Academic Press.

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EXPRESSION ANALYSIS OF SMARC NUCLEIC ACIDS AND POLYPEPTIDES USEFUL IN THE DIAGNOSIS AND TREATMENT OF PROSTATE CANCER; EVALUATING HUMAN FOR PROSTATE CANCER; OBTAIN SAMPLE, MONITOR PREFERENTIAL GENE EXPRESION, COMPARE TO CONTROL, AMPLIFIED GENE EXPRESSION IS INDICATIVE OF PROSTATE CANCER

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Abstract: The invention relates to compositions, kits, and methods for detecting, characterizing, preventing, and treating prostate cancer. SWI/SNF-related matrix-associated actin-dependent regulator of chromatin (SMARC) markers are provided, wherein changes in the levels of expression of one or more of the SMARC markers is correlated with the presence of prostate cancer.

Non-exemplary Claims: ...of a therapy for inhibiting prostate cancer in a subject, comprising: a) expression of a **SMARCD3** marker in a first sample obtained from said subject prior to providing at least a portion of said therapy to said subject; b) expression of the **SMARCD3** marker in a second sample obtained from said subject following therapeutic treatment; and c) comparing...

...in a subject at risk for developing prostate cancer, the method comprising inhibiting expression of **SMARCD3** marker in the cells of a subject **SMARCD3** marker in a cell in the presence of a test compound; and b) comparing the expression measured in step (a) to the expression of the **SMARCD3** marker in a cell in the absence of said test compound, wherein said compound is...

...compound, wherein said compound is useful for treating prostate cancer

when the activity of the **SMARCD3** marker in the presence of said test compound is higher than its activity in the...

...administering to the patient in need thereof a compound which increases the expression of a **SMARCD3** marker...

...31. The method of claim 30, wherein said compound increases the expression of a **SMARCD3** mRNA...

...32. The method of claim 31, wherein said compound decreases expression of a **SMARCD3** marker protein...

...in said subject sample at a first point in time, the expression level of a **SMARCD3** marker; b) repeating step (a) at a subsequent point in time occurring after said subject...

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